CLINICAL PROFILE OF 50 CASES OF ATRIAL FIBRILLATION

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CERTIFICATE

This is to certify that the dissertation titled **"CLINICAL PROFILE OF 50 CASES OF ATRIAL FIBRILLATION"** is the bonafide original work of DR. P. V. GIRISH in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in February 2006. The Period of study was from January 2004 to January 2005.

PROF. S. NATARAJAN, M.D. Professor and Head of the Dept. of Medicine, Govt. Stanley Medical College and Hospital Chennai-600 001.	**PROF. T. VENKATAKRISHNAN** Addl. Professor of Medicine Govt. Stanley Medical College and Hospital Chennai-600 001.

DEAN
Govt. Stanley Medical College & Hospital,
Chennai – 600 001.

DECLARATION

I, **DR. P.V. GIRISH,** solemnly declare that dissertation titled **"CLINICAL PROFILE OF 50 CASES OF ATRIAL FIBRILLATION"** is a bonafide work done by me at Govt. Stanley Medical College and Hospital during January 2004 to 2005 under guidance and supervision of my unit chief **Prof. T. VENKATAKRISHNAN**., Addl. Professor of Medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place : Chennai.

Date :

(Dr. P.V. GIRISH)

CERTIFICATE OF EXPERIENCE

To whomsoever it may Concern. This is to certify that **Mr. K. ARULMOORTHY**, is working in our concern since **June 2003 to August 2005** as **Site Engineer**. He is experienced in Site Execution, Labour Management and his character is **Good**.

Thanking you,

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INTRODUCTION

Atrial Fibrillation

Atrial fibrillation (AF) is the commonest cardiac arrhythmia, and is increasing in frequency. Atrial fibrillation is an abnormal heart rhythm during which the upper chambers of the heart beat irregularly. Normally the pacemaker of the heart generates an electrical impulse, which is conducted or carried to the lower or pumping chambers of the heart via the electrical conducting tissues of the heart. This allows a natural sequence of contraction where the upper chambers (atria) beat first, thus filling the lower chambers (ventricles). This sequence allows priming of the pumping chambers and contributes as much as 20% of the output of the heart. In atrial fibrillation, the heart's natural pacemaker, the sinus node, no longer generates an electrical impulse. Instead electrical activity occurs irregularly throughout both left and right atria. This irregular electrical impulse is conducted erratically to the ventricles, resulting in an irregular heartbeat which may be excessively fast and vary in volume from beat to beat. The atrial electrical activity is very rapid (approximately 400 to 700 beats/min), but each electrical impulse results in the depolarization of only a small islet of atrial myocardium rather than the whole atrium. As a result, there is no contraction of the atria as a whole. Since there is no uniform atrial depolarization, there is no P wave. The chaotic electrical activity produces deflection on the ECG, referred to as fibrillatory wave.

Fibrillatory waves vary in size and shape and are irregular in rhythm. Transmission of these multiple atrial impulses into the AV node is thought to occur at random, resulting in an irregular ventricular rhythm. Some impulses are conducted into, but not through, the AV node; i.e., they are blocked within the AV node. This is a form of "concealed conduction" and is important since such non-conducted impulses contribute to the overall refractoriness of the AV node. For this reason, the ventricular rate of atrial fibrillation is often slower (averaging 160 to 180 beats/min)

Today, more than 5% of those over 65 have AF. And most of these cases have underlying structural heart disease, which may be hypertensive, ischemic, or valvular in nature. The risk factors for AF include: male sex, hypertension, diabetes, thyrotoxicosis, heart failure, valvular disease, and excess alcohol intake. Rarely, AF occurs in a familial pattern; it's probable that more than one gene is involved.

The chief consequence of AF is an increased likelihood of stroke; this occurs in 1.5% of persons with AF in their 50s, to a 23.5% risk for those in their 80s. Mortality rates are doubled, in both sexes, due to stroke, heart failure, or myocardial infarction.

When in atrial fibrillation the patient may feel his/her heart beating rapidly and irregularly (palpitation). Atrial fibrillation may cause chest pain, shortness of

breath, dizziness, weakness or fatigue. In some patients there are no accompanying symptoms.

If atrial fibrillation causes chest pain, shortness of breath, dizziness or congestive heart failure, the arrhythmia may be dangerous and need to be corrected promptly. Usually however symptoms are not that severe and the arrhythmia may be dealt with less acutely. The major long-term danger of atrial fibrillation is an increased risk of congestive cardiac failure and stroke. The atria of the heart do not contract properly during atrial fibrillation. Blood flow is sluggish within the atria and this may lead to clot formation. If one of these clots breaks loose, it may travel to other parts of the body (embolism) resulting in stroke (cerebral embolism) or blockage of blood vessels throughout the body. Blockage of vessels to the limbs may cause inadequate blood supply (ischaemia) and endanger the limb. Similarly, blockage of a blood vessel in the abdomen may cause abdominal pain and bowel ischaemia, a condition that is life threatening.

A typical ECG in atrial fibrillation shows a rapid irregular tachycardia in which recognizable P waves are absent. QRS complexes are generally normal, and the ventricular rate in patients with untreated atrial fibrillation generally ranges between 150 and 220 beats/min. However, in elderly patients, ventricular rates in untreated atrial fibrillation are typically slower. The ventricular rate may be accelerated in the presence of thyrotoxicosis, fever, catecholamines or catecholamine-like drugs, or conditions that enhance sympathetic tone. Although a

cardinal feature of atrial fibrillation is irregularity of the RR interval, at the most rapid ventricular rate, this irregularity may be somewhat difficult to discern. An increasing incidence and morbidity due to atrial fibrillation prompted to study the patients with atrial fibrillation in our institution.

AIM OF THE STUDY

1. To Study age, sex distribution and etiologic analysis of atrial fibrillation
2. To study the symptom profile of atrial fibrillation
3. To assess left atrial size and relation of that to permanent atrial fibrillation
4. To assess the incidence of Left atrial clot in cases of atrial fibrillation by Transesophageal Echo

REVIEW OF LITERATURE

History :

Evaluation of the peripheral pulse has fascinated physicians for centuries. Moses maimonides around 1187 described an irregular pulse. Prior to the electrocardiographic confirmation of atrial fibrillation, many observant physicians commented on grossly irregular pulses that were likely to be atrial fibrillation.

In 1904 Wenckebech published monograph on cardiac arrhythmias he described excessive irregularity or “delirium cordis”.

Csuhny, Mackenzie, Rother Berger and Winter Beg especially Lawis made significant early observations concerning atrial fibrillation. The pioneering work of Einthores enabled clinical investigators to record the electrographic representation of the clinical observation of atrial fibrillation.

Pathology:

The incidence of atrial fibrillation increase with age and it is especially prevalent in those aged 60 or greater. Macroscopic and Microscopic alternations in the atrial tissue begin in the first year of life. By fourth and fifth decades small fat spots appear in the right atrium in the region of AV node and septum. These changes associated with ageing will result in loss of myocardial fibers and increase in fatty metamorphosis of connective tissue and focal hemorrhages in the sinus node, AV node and atrial structures. One of the most important pathological

studies of atria in patients with atrial fibrillation was done by Davies and Pomerance. In nearly 75 percentage of patients with chronic atrial fibrillation there was sinus node muscle loss, internodel tract muscle loss and atrial dilation.

In case of rheumatic heart disease the left atrium is enlarged in almost all cases[18,20]. In some cases of tight mitral stenosis Aschoff bodies are found with in the atrial myocardium. In other cases spindle shaped or triangular scar lying between the muscle bundles and surrounding blood vessels represent the healing of the Aschoff bodies

MECHANISM OF ATRIAL FIBRILLATION

The two chamber concept

In an electrophysiological sense the heart consists of only two chambers one formed by the atria and the other formed by the ventricles.

The two electrophysiological chambers are separated from each other by an electrical conduction barriers formed by the fibrous AV Ring. Communication across these barriers under normal circumstances is only possible through the specialized conduction system formed by AV node, the bundle of His, the bundle branches and their ramifications.

Each chambers is activated by single, coordinated, uniform and pro gressive excitation process which effects a total and almost simultaneous depolarization's of the chamber. All the fibers are rapidly brought to the same state of excitation.

Consequently the fibers of the chambers will at any given moment be in the same or almost the same electrophysiological state. They will all be in a state of excitation or all in a state of potential responsiveness and when all the fibers of a chamber are uniformly in one state, they are said to be in phase with each other. In myocardial fibrillation the single uniform activation process is lost and the in phase state is transformed into a complex out of phase state. A physiological fragmentation tissue is less in varying states of refractoriness, excitation and responsiveness occur.

The pre disposition to any development of this state favored by the co incidence of two fundamental events:

1. Uneven recovery of the chambers an out of phase state where one part of the chamber is responsive and another part of the chamber is refractory, a state of physiological asymmetry.
2. Premature stimulation of chamber by an impulse that originates in or is introduced into the chamber before activation or recovery is complete.

The Genesis of Physiological Asymmetry.

The development of Physiological asymmetry with in the bi-atrial chamber is favored by:

a. The mode of atrial activation longitudinal activation.
b. Prolongation of conduction.
c. Unequal conduction.

d. Increase in chamber size.

e. The chamber asymmetry.

f. Abbreviation in refractoriness

g. Unequal refractoriness.

A. The mode of Atrial activation:

The most important factor predisposing to the development of atrial activation.

Activation of the bi-atrial chamber occurs longitudinally and by contiguity spreading from its point of origin to engulf the whole chamber each fiber in turn activating the adjacent fibers. The initial and terminal discrepancies resulting from longitudinal activation will in turn be affected by and depend upon factors such as the size and shape of the bi atrial chamber, the conduction time and the duration of the refractory period.

B. Prolongation of Conduction:

A prolongation of conduction time will be associated with longitudinal activation, increasing the initial and terminal discrepancies.

C. **Unequal Conduction:**

Unequal conduction within the bi-atrial chamber will also potentiate simple out of phase state due to differences in intramural tension and disease process such as local ischemia.

D. Increase in chamber size:

The larger the size of the chamber the greater the potential simple out of phase state and more readily will fibrillation be initiated and maintained. Atrial fibrillation is especially common in cases of mitral and tricuspid incompetence conditions which are usually associated with massive atrial enlargement.The AFFIRM[3] study noted that two thirds of patients with atrial fibrillation had siginificant left atrial dilatation. Adbou Elhendy et al[20] in his study had repoted left atrial dilatation in 80% of AF of all causes.

E. **The chamber Asymmetry:**

The bi atrial chamber is asymmetrical and the pacemaker is eccentrically placed. Consequently activation and recovery occurs sooner at a point close to the pace maker than at a point remote from the pacemaker. The bizarre or asymmetrical shape of the bi atrial chamber will therefore, in the presence of longitudinal activation predispose towards the unequal recovery of the chamber.

F. **Abbreviation of refractoriness:**

A short refractory period when associated with a long or relatively long conduction times will also predispose to the maintenance of fibrillation.

G. Unequal refractoriness:

Unequal refractoriness of the bi-atrial chamber will also aggregate the terminal out of phase discrepancy and predispose to atrial fibrillation.

The maintenance of Atrial Fibrillation

The fibrillation will not be maintained unless there is a relatively short refractory period of parts of the myocardium and is associated with a long or relatively long conduction times.

Any condition which shortens the refractory period will facilitate the perpetuation of fibrillation.

Following conditions shortens the refractory period:

1. Acetycholine
2. Vagal Stimulation
3. Very low concentration of potassium
4. High concentration of potassium
5. Digitalis in toxic doses

All these factors facilitate the precipitation and perpetuation of fibrillation. Atrial Fibrillation is mostly maintained in cases of tricuspid and mitral regurgitation that are associated with large atria.

Causes of Premature atrial Stimulation:

The development of an out of phase state with in the bi atrial chamber predisposes it to the precipitation of fibrillation by pre mature stimuli.

There are four Basic Sources of such premature stimuli:

1. Atrial extra systoles.
2. Atrial tachycardia.
3. Reciprocal stimuli.
4. A circus movement stimulus.

1. Atrial extra systoles:

Atrial extra systoles are pre mature impulses which arise from an ectopic atrial pace maker and these particularly will favor the initiation of fibrillation.

2. Atrial Tachy Cardia:

Tachycardia shortens the refractory periods but with very fast rates, the refractory periods is reduced to a critical level when it cannot shorten any further, it occupies virtually the complete diastolic interval. When this occurs, successive impulse will tend to fall in the terminal out of phase period and may there by initiate fibrillation.

3. Reciprocal Stimuli:

Pre mature stimulation of atria is also facilitated by the presence of a by pass with in the AV conduction pathway. The rapidly returning reciprocal impulse constitutes an early stimulus to the bi atrial chambers and may consequently initiate the fibrillation in the WPW syndrome and LGL syndromes.

4. A circus movement stimulus:

A circus movement is a from of activation where in the excitation wave travels in a circular path around the bi atrial chamber. The phenomenon occurs when the time taken for the excitation wave travel around the bi atrial chamber exceeds the refractory period. .

The mechanism is facilitated by the association of a long conduction time and a short refectory period. A circus movement may be responsible mechanism in some cases of atrial flutter.

Atrial flutter frequency precedes the atrial fibrillation. The flutter constitutes a source of early stimulation by virtue of a circus movement of rapid repetitive stimulation and the flutter rate frequently increase just before the conversion to atrial fibrillation.

A circus movement , reciprocal mechanism, atrial extra systoles, and atrialtachycardia are in the contest for basic mechanisms, but merely constitute sources of pre mature and or rapid stimulation.

The aforementioned principles indicate that in the presence of longitudinal activation the initation of fibrillation is favored by enlargement of the bi atrial chamber, prolongation of conductions inequality of conduction, diminution of refractoriness and unequal refractoriness.

The mechanism of atrial fibrillation was the multiple hypothesis of Moe[39]. He noted, “The grossly irregular wave front becomes fractionated as it divides

about islets or strands of refractory tissue, and each daughter wavelets may now he considered as independent off spring such a wavelet may accelerate or decelerate as it encounters tissue in a more or less advanced state of new range" . Thus more such wavelets present the more likely the arrhythmia will sustain. The numbers of wavelets depends on the atrial mass, refractory period and conduction velocity of various areas of the atria. In essence a large atrial mass with short refractory periods and conduction delay would yield increased wavelets and would be most favorable for sustaining atrial fibrillation. Recent observations on the initiation of AF from area of pulmonary veins[40] & concept of fibrillatory conduit and rotors have rekindled the consideration of some older concepts. Atrial remodeling is considered to be an important determinant of the Pathophysiology[40] where the modulating factors are endothelial dysfunction, hormonal, inflammatory mediators like Crp,IL-6.

Electrocardiographic manifestations of Atrial Fibrillation.

Atrial Fibrillation is characterized by the following electro cardiographic features.

1. Irregular bizarre and chaotic atrial deflection – "F" waves which distort the base line.
2. An irregular AV conduction sequences resulting in a completely irregular arrhythmia with variation in RR interval.

The atrial deflections.

Atrial activation is chaotic and virtually a self perpetuating process. The atrial deflections are bizarre, irregular, chaotic and recorded contiguously and the atrial activation is instead reflected by variable, Irregularly corrugated, deflections which continuously deform the baseline. These are inscribed at an irregular rate which raise from 400 to 600 waves / mt.

When “F” wave deflections are large in amplitude and low in frequency the condition is called; Atrial Fibrillation. When these waves are rapid and are of small amplitude (< 1mm) the term “Fine” Atrial Fibrillation used. In long standing atrial fibrillation. the fibrillatory waves may disappear leaving an almost smooth baseline is formed resembling atrial stand still.

Some time regular flutter like large waves are seen for a few second followed by smaller irregular waves typical of Atrial Fibrillation.

This condition is called impure flutter fibrillation.

AV Conduction

Conduction of impulses from the fibrillating atria may be complicated by

a. Physiological second degree AV block
b. High grade AV block
c. Complete AV block

Atrial Fibrillation with physiological degree AV block.

Here AV node offers block because of its inherent refractory period when AV node is bombard with a very large number of stimulus from the atria, which may fall on the refractory period of the AV node are blocked concealed conduction and conduction occurs only when the node recovers. Because of the extreme irregularity of the atrial impulses the block also occurs irregularly and so the QRS complexes are placed at irregular intervals. In this type of block the ventricular rate is usually between into beats / minute.

At times the distributional pattern of the QRS complexes in atrial fibrillation suggests a Wenkeback form of AV conduction. This is suggested by a progressive diminution in RR intervals followed by a long pause.

ATRIAL FIBRILLATION WITH HIGH GRADE AV BLOCK

This is characterized by idiondal or idiventricular escape beats that occurs regularly. Here the longer intervals of QRS complexes the fibrillation remains constant.

Atrial Fibrillation with Complete Heart Block

This is characterized by slow and regular ventricular rate. The beats are due to idionodal or idioventricular rhythm. In the former rhythm the QRS complexes are narrow while in the later the complexes are wide and have bizarre shapes.

Intra Ventricular conditions:

This may be normal or complicated by phasic aberrant conduction. The phasic aberrations are temporary abnormalities in the ventricular conduction of supra ventricular impulses when impulses from above reach the ventricles at a rapid successions some times a QRS patterns is produced. The successive impulses which reach the ventricles with sufficiently longs intervals produce normal QRS complexes. These aberrant complexes are not attended by compensatory passes.

Type of Atrial Fibrillation Based on:

1. Ventricular Activity:

Fast Atrial Fibrillation when the ventricular rate is more then 120 beats / mt.

Slow atrial fibrillation: When the QRS complexes occur at a slow, rate 60-120 / min

Both these terms are misnomers since the atrial rate is very high in both cases and the term fast and slow here applies to the ventricular rate.

Atrial Fibrillation with regular ventricular rate:

This occurs:

a. In complete heart block with idionodal or idioventricular rhythm.
b. After administration of drugs like verapamil.
c. Junctional or ventricular tachycardia.

2. Duration:

PAROXYSMAL ATRIAL FIBRILLATION : A paroxysm of atrial fibrillation lasting less them 48 hrs. This may be seen in normal subjects particularly during emotional stress on following surgery, exercise, acute alcoholic intoxication or a prominent surge of vagal tone. It may also occur in patients with heart or lung disease who develop acute hypoxia, hyperpnea, metabolic or hemodynamic derangements[45].

Persistent Atrial Fibrillation:

An episode of atrial fibrillation lasting greater than 48 hrs, which can still he converted to sinus rhythm. Usually occurs in patients with cardio vascular disease, most commonly rheumatic heart disease, hypertensive cardio vascular disease, chronic lung disease, atrial septal defect any variety of miscellaneous cardiac abnormalities.

Permanent Atrial Fibrillation:

Inability of pharmacologic or non pharmacologic methods to restore sinus rhythm.

So called lone Atrial Fibrillation which occurs in patients with out underlying heart disease often represents the tachy cardiac phase of the Tachy cardiac – Brady cardiac syndrome.

Etiology of Atrial Fibrillation:

Essentially cardio vascular diseases:

1. Rheumatic Heart Disease
2. Coronary artery Heart Disease
3. Hypertensive Heart Disease
4. Cardiomyopathy
    - Dilated
    - Hypertrophic
5. Congenital Heart Disease
    - ASD
    - Lutem Bacher’s syndrome
    - Tricuspid atresia
    - Ebstein anomaly
    - Corrected transport ion of great arteries
6. Sick Sinus Syndrome
7. Diffuse myocardial disease; myocarditis
8. Pericardial Disease
    - Constrictive pericarditis
    - Pericardial effusion

9. Pre excitation syndromes
    - WPW syndrome
    - LGL syndrome
10. Syphilitic Heart Disease
11. Bacterial Endocarditis
12. Atrial Mycenae
13. Pulmonary heart disease
14. Cardiac Surgery

    -Cardiac vascular surgery

    - Coronary artery surgery

II Metabolic Diseases.

1. Hyperthyroidism
2. Hypothyroidism
3. Hypokalemia
4. Uremia

III Intra Thoracic causes

1. Pulmonary Embolism
2. Labor Pneumonia
3. After pneumonectomy
4. Chronic lung disease
5. Intra thoracic tumors infiltrating the heart.

IV Neurologic Disease

1. Meningitis
2. Meniere's Syndrome
3. Following Head Injury
4. Emotional stress

V Miscellaneous Causes

1. During Anaesthesia
2. Drugs and chemicals
    a. Alcohol
    b. Smoking
    c. Coffee
    d. Digitalis
    e. Adernaline
3. Vomiting Spells
4. Extreme exercise
5. pregnancy

VI Lone Atrial Fibrillation

Idiopathic

RHEUMATIC VALVULAR HEART DISEASE:

Cases of mitral and tricuspid valvular disease are frequently associated with atrial fibrillation because these conditions have the following pre disposing factors:

a. Enlargement of the atria
b. Prolongation of the Atrial conduction time
c. Differential refractoriness of the atrial myocardium
d. Atrial Extra systoles

Rheumatic Valve disease especially is particularly important as it increases the thromboembolic risk of patients with chronic atrial fibrillation about 5 fold, up to a fifty% of patients with mitral valve stenosis and atrial fibrillation develop embolic events which in most cases affect the cerebral circulation. Echocardiography in such cases may show left atrial thrombus. Trans esophageal echocardiography is more sensitive than transthoracic echocardiography[21,42]. Kaymaz et al[21] had discerned the characteristics of left atrial thrombus predisposing to systemic embolism with transesophageal echocardiogram.

Coronary Artery Disease:

Ischemic heart disease is probably the most common underlying cause of atrial fibrillation in western countries. In addition the fast ventricular rate due to atrial fibrillation may cause angina, leading to cardiac ischemia and heart failure

atrial fibrillation may complicate acute myocardial infarction in 10 to 15% of cases and is often a marker of extensive myocardial damage and poor prognosis with increased mortality. It atrial fibrillation occurs with an acute myocardial infraction it tends to occur in the first 24 hrs and is usually self limiting.

Atrial fibrillation is also a marker of underlying ventricular dysfunction and a compromised myocardium. Many years after myocardial infarction, ventricular scarring and dilatation often predispose to atrial fibrillation and congestive cardiac failure.

Hypertensive Heart Disease:

Hypertension accounted for about half of the cases of atrial fibrillation in the Framingham study. Hypertension contributes to the complication of stroke and thromboembolism in such patients, especially if left ventricular hypertrophy is present.

Atrial fibrillation may be secondary to left atrial dilatation which occurs in hypertensive patients as a consequence of reduced left ventricular compliance. In addition hypertension may be associated with underlying coronary artery disease, which itself is a risk for atrial fibrillation and thromboembolism.

Congestive Cardiac Failure:

Congestive cardiac failure from any cause may predispose to atrial fibrillation this is favored by associated enlarged atria and the consequent distension and stretch of the atrial walls.

Congenital Heart Disease:

Atrial Fibrillation is common is two form of congenital heart diseases atrial septal defect and Ebstein anomaly. In ASD the incidence of atrial fibrillation begin to increase starting from the fourth decade of life. Epstein's anomaly is commonly associated with atrial fibrillation often at an early age.

Cardiac Surgery:

Atrial Fibrillation is the most frequent arrhythmia noted after cardiac surgery patients undergoing coronary artery by pass graft surgery one of the largest group to have first episode of post operative Atrial Fibrillation.

Post operative discontinuation of Beta Blocker's taken regularly before surgery increases the risk of atrial fibrillation.

Increased post operative sympathetic activity demonstrated by elevated norepinephrine levels is associated with increased risk of atrial fibrillation, minimally invasive cardiac surgery may not reduce the incidence of post operative atrial fibrillation.

WPW – Syndrome:

In WPW syndrome patients, an atrial dysarrythmia associated with an ante grade conducting accessory pathway can be life threatening. Aberrant conduction usually occurs when a short RR interval follows a long RR interval (Ashman's Phenomenon). In those patients AV node blocking agents such a digitalis beta blockers or calcium channel blockers are contra indicated because they cause

ventricular fibrillation. Intravenous procainamide or lidocaine can be given. Radiofrequency ablation is the treatment of choice.

Hypertrophic Cardiomyopathy:

Approximately 15% of patients with hypertrophic cardiomyopathy develop atrial fibrillation. In these individuals atrial fibrillation results in profound hemodynamic disturbance resulting in chest pain, presyncope, syncope, and even sudden cardiac death.

Tachy Cardia Induced Tachycardia:

Tachycardia induced tachycardia is a phenomenon in which one tachy cardiac degenerates in to another tachycardia. A relative common example is rapid ventricular tachy cardiac that degenerates in to ventricular fibrillation. Atrial tachycardia and atrial flutter are likely to be the most common cause of tachycardia induced tachycardia resulting in atrial fibrillation.

Hyperthyroidism

Atrial Fibrillation is not uncommon in thyrotoxicosis. Thyrotoxicosis is an important curable cause of atrial fibrillation .About 10 to 15 percent of patients with untreated thyrotoxicosis develop atrial fibrillation, it is more frequent in men and increase with age.

Thyroid hormone affects the circulatory system and myocardium. There is decrease in total systemic vascular resistance, an increase in total blood volume and decreased circulation time this leads to an increase in preloads and reduction

in after load. There is also reduction in electrical threshold for excitation of atrium and increase in sinus rate.

There appears to be an interaction between excess thyroid hormone and catecholamine action, including potentiation of catecholamine effects and increased number of cardiac beta adrenergic receptors.

Thyrotoxicosis may be under diagnosed particularly in elderly people in whom classic signs of thyrotoxicosis may not be obvious. One clinical clue may be the failure of digoxin to control the ventricular rate without the addition of betablockers. Hyperthyroidism may coexist with both ischemic and rheumatic heart disease.

Neurogenic Cause of Atrial Fibrillation:

The sustained Atrial Fibrillation is facilitated by increased para symphathetic tone. Atrial refractory period is decreased by acetylcholine which shortens the wave length making it easier for Atrial Fibrillation to sustain. Atrial Fibrillation occurs during enhanced vagal tone for example onset of atrial fibrillation during swallowing cold substance, ice cream.

Adrenergic form of Atrial Fibrillation has also been described. The characteristics include

a. On set during day time
b. Preceded by emotional stimulus of exercise
c. Polyuria common
d. Atrial Fibrillation

With specific sinus rate. Beta-blockers are the treatment or choice.

Excess alcohol intake:

Atrial Fibrillation due to an excess in take of alcohol often occurs after holidays or at week ends giving rise to the term " Holiday Heart syndrome". Alcohol can be associated with a dilated heart (alcoholic heart muscle disease) and atrial fibrillation.

Pneumonia:

Pneumonia is commonly associated with atrial fibrillation. In about seven percent of cases, pneumonia as a precipitant of atrial fibrillation occurs pre dominantly in elderly patients.

Idiopathic or Lone Atrial Fibrillation:

Some patients with atrial fibrillation have no predisposing factor or cardiac lesion. The condition in these patients is classified as "lone or idiopatic" Atrial Fibrillation.

Familial Atrial Fibrillation:

Familial atrial fibrillation is extremely uncommon seen relatively in young age less than 20years old. The gene responsible for atrial fibrillation was located on chromosome 10g in the region of $10g_{22}$ to $10g_{24}$.

Clinical Features of Atrial Fibrillation

Symptoms:

Palpitation

Dyspnoea

Fatigue

Angina

Presyncope

Syncope

Atrial Fibrillation is associated with discrete group of symptoms. Palpitation being the most common. Other frequent symptoms are dyspnea, fatigue and pre syncope and less common symptoms were chest pain and syncope. In a small group of patients atrial fibrillation as no symptoms.According to the Canadian Registry of Atrial fibrillation(CARAF) one fifth of patients were asymptomatic & palpitation was the leading symptom.

Physical findings:

Pulse is irregularly irregular. Absence of “a” waves in the jugular venous pulse, results in a single positive wave form .

With fast ventricular rates an apex-radial pulse deficit appears as each contraction may not be sufficiently strong to transmit an atrial pulse wave through the peripheral artery.

The blood pressure reading depends upon the cause of atrial fibrillation and the cardiac status although the pulse pressure is generally variable.

Cardiac auscultation will reveal the variable intensity of the first heart sound because of the fluctuation in ventricular filling periods and the signs of underlying heart disease like mitral stenosis.

COMPLICATIONS:

Heart Failure:

The sudden onset of fast atrial fibrillation may often precipitates overt heart failure particularly if left ventricular function is already compromised by coexisting heart disease for example valvular or ischemic heart disease.

Heart disease is associated with atrial fibrillation in about 35 percent of cases. In these patients atrial fibrillations may be a marker of increased mortality and may also enhance the substantial risk of thromboembolism.

Thromboembolism:

Atrial Fibrillation predisposes to the formation of intracardiac thrombus, which may result in stroke and thromboembolism. The commonest site of thrombus is the left atrial appendage. Right atrial thrombus with subsequent pulmonary thromboembolism is a rare complication. The Framingham heart study[4] correlated increased risk of stroke and death with increase in left atrial size. Goswami K.C et al[41] reported 25% incidence of LA appendage clot and 50% incidence of spontaneous echo contrast with transesophageal echo study on 200 patients with rheumatic mitral stenosis and af. Srimannarayanan[44] J reported LA clots in one third of patients with severe MS and AF in their study on 490patients

and that LA appendage clots disappear with anticoagulants where as those extending to left atrial body may persist despite optimal anticoagulation.

Stroke:

The risk of stroke in some one with atrial fibrillation is about five percent a year, and epidemiological evidence suggest that the risk increase with age, raised blood pressure and other evidence of heart disease. Non rheumatic atrial fibrillation increased five fold the risk of stroke[46]. Patients with atrial fibrillation may also have an increased risk of recurrent stroke and silent cerebral infarcts (often multiple) on computed tomography. Patients with acute stroke and atrial fibrillation have a significant higher mortality than patients in sinus rhythm (23% vs 18% in the oxford community stroke project). The higher mortality is explained partly by the association of atrial fibrillation with large, total anterior cerebral infarcts, probably due to occlusion of the middle cerebral artery.

DIFFERENTIAL DIAGNOSIS:

Atrial extrasystoles:

Atrial extrasystoles occur commonly and may account for an irregular pulse leading to atrial fibrillation being wrongly diagnosed, long pauses may follow as sinus node automaticity is depressed by the extra systole. Multifocal extrasystoles are particularly common in pulmonary disease.

Atrial Tachycardia:

Atrial Tachycardia may simulate atrial fibrillation with rapid ventricular rate, “P” wave precede each QRS complex in atrial tachycardia, RR interval is perfect. Carotid sinus massage may terminate a tachycardia and produce a regular and normal rate.

Atrial Flutter:

At times the rhytm may alternate between flutter and fibrillation in a single tracing. There are border line cases where precise differentiation cannot be made (Flutter Fibrillation). Atrial flutter and fibrillation form the extreme of the same spectrum of arrhythmias.

Flutter waves are seen as regular saw tooth like atrial activity most prominent in the inferior leads.

Atrial flutter is characterized by atrial rate between 250 and 350 beats / miutes typically, the ventricular rate is half. The ventricular rate in 2:1 AV block is approximately 150 bpm. If the atrial rate slowed to less than 220 beats per minute the ventricular rate may rise suddenly because of the development of 1:1 AV conduction.

INVESTIGATIONS:

Electrocardiography:

The arrhythmias should be documented firstly with a conventional 12 lead ECG. This may provide a clue to the etiology or electrophysiological features that may cause arrhythmia for example ischemic heart disease, left ventricular hypertrophy or a preexitation syndrome.

It was thought that coarse fibrillatory waves favour the etiology of rheumatic mitral valve disease or hyperthyroidism while five fibrillatory waves are associated with atherosclerotic or hypertensive cardio – vascular disease (Goldman 1979). This view is contradicted in recent studies.

A 24 hour hotter monitor may be needed to document paroxysmal atrial fibrillation or sick sinus syndrome.

Chest Radiography:

A chest radiography is useful in most cases of atrial fibrillation. In a young patient it may provide a clue to congenital heart disease. Such as ASD. X-Ray film can give information regarding the size of the heart and whether the patient has heart failure. Barium swallows in appropriate cases show displacement of the esophagus.

Blood Tests:

Full blood counts – especially when anti coagulants are being considered.

Urea and electrolytes - To establish baseline and if considering drug treatment.

Thyroid function tests in suspected cases of hyperthyroidism.

Rheumatic heart disease – ESR, ASO titres.

Myocardial infarction – enzymes studies CPK – MB, Troponin ‘T’.

Echocardiography:

An echo cardiogram is an important test to obtain in patients with atrial fibrillation. It allows the evaluation of atrial size, right and left ventricular function, presence of congenital and valvular lesions.

Transesophageal echocardiography is valuable to investigate atrial function in patients with atrial fibrillation. It provides information of the presence of thrombi prior to cardioversion, if such a procedure in contemplated.

Exercise Testing:

Exercise testing in necessary in some patients with IHD’s and atrial fibrillation to clarify the severity of underlying cardiac ischemia. Exercise electrocardiography must be interpreted cautiously, how ever if treatment with digoxin is given such testing may also have a role in assessing the adequacy of drug treatment in controlling the ventricular response in atrial fibrillation.

Electrophysiology Studies:

In patients with atrial fibrillation due to pre-excitation syndromes, electrophysiology studies may be needed to document the characteristics of conduction from the atria to the ventricles and the presence of accessory pathways. This may lead to a “Cure” of the condition by transcatheter ablation of the accessory pathway.

MATERIAL AND METHODS:

This study was conducted at Govt. Stanley Medical College & Hospital, Chennai. This study was conducted during the period of one year Jan 2004-December 2004, fifty cases of atrial fibrillation were included in this study. No patient had been counted twice if he or she got admitted again after discharge during the period.

The Diagnosis of Atrial Fibrillation:

This is made on clinical grounds and then confirmed by electro cardiographic methods.

Clinical Grounds:

1. Irregularly irregular pulse : If the patient was not in failure he or she was exercised and the persistence of the irregularity is noted.
2. Pulse deficit simultaneous counting of the pulse rates by on observer and the heart rate by another of one full minute.
3. Absence of ‘a’ wave in the jugular venous pulsation, in areas where jugular venous pulsation is seen.
4. On auscultation varying intensity of the first heart sound.
5. Carotid sinus massage once again the irregularity persists though the heart rate is slowed

E.C.G Recording

A 12 lead electrocardiograph was taken for all the cases. It was standardized to produce a deflection of 10mm per 1mv input and the paper speed was set at 25mm per sound. The ECG features of atrial fibrillation are

1. Absence of regular rhythmic ‘P’ were
2. Atrial activity is reflected by a variable irregularly corrugated deflection deforming the base line ‘F’ wave.
3. Marked variation in RR Interval
4. Variation in QRS complex configuration in ECG

Other findings like left ventricular enlargement right ventricular enlargement, right bundle branch block, evidence of ischemia and infarction were also looked.

In all cases, complete history was taken and general examination was done. Further points were noted according to the suspected etiology.

Rheumatic Heart Disease:

1. Features of Rheumatic fever (as per the updated Jones criteria published in 1992, by the American Heart Association)
2. Features of congestive cardiac failure (as per the Framingham criteria, circulation 88:107, 1993).
3. The presence of valvular heart disease
4. Features of infective endocarditis

5. E.C.G for ventricular hypertrophy patterns apart from Atrial Fibrillation.
6. Plain X-Ray of PA view.

Coronary Heart Disease:

1. History
2. Peripheral Arterial Thickening
3. Auscultation for S_3 and S_4 (which may denote reduce compliance of the ventricles)
4. Study of ocular funds.
5. X-Ray for cardiomegaly, pulmonary congestion
6. Serum enzymes and cholesterol.

Hypertensive Heart Disease:

Blood pressure, fundus examinations, E.C.G for left ventricular hypertrophy, X-Ray chest for cardiomegaly, Urine analysis, Blood urea level, if necessary other investigation to find out whether the hypertension is primary or secondary.

Hyperthyroidism:

Eye Signs

Tremors

Thyromegaly

Sleeping pulse rate

E.C.G

T_3, T_4, TSH with the help of private laboratories.

Chest Radiography :

A Posteroanterior view of the chest radiography was taken and evaluated for evidences of valvular heart disease, congenital heart disease, pericardial effusion chronic obstructive pulmonary disease, pneumonia etc.,

Echocardiography :

M – Mode, 2-D, Echocardiography was done for all the patients. The rhythm of the heart was looked in to. The presence of thickening of valves, calcification and valve closure was noted, the size of the valve rings and chambers of the heart was assessed. The appendages were specially looked and vegetations also noted. Atrial enlargement , ventricular function also noted. They were also subjected to transesophageal echo for better characterization of LA clot[42] as TEE is superior to TTE for the same. Under local anaesthetic spray, gastroscope like instrument with ultrasound probe was introduced and views taken[45].

Diagnosis of complications

Atrial Fibrillation complicating cardiac failure patients, complete history and examination were done. Electrocardiographic features, chest radiography for cardiomegaly and radiography for cardiomegaly and pulmonary congestion, echocardiographic evaluation of ventricular function were noted.

Atrial fibrillation complicating stroke, patients neurological examination done. CT scan brain taken and the features were noted. Stroke patients associated with atrial fibrillation the presence of thrombus in transesophageal echocardiogram also noted.

In atrial fibrillation patients features of infective endocarditis clinically evaluated. In suspected patients three samples of blood culture taken one hour interval, and result noted. Presence of vegetations in the echo cardiography also noted.

RESULTS

TABLE 1

AGE DISTRIBUTION

Age in Years	No. of Patients	Percentage
11-20	8	16%
21-30	7	14%
31-40	8	16%
41-50	15	30%
51-60	8	16%
61-70	4	8%
Total	50	100%

The mean age was found to be 43 years. Most of the patients belong to age group of 31-50 years.

TABLE 2

SEX DISTRIBUTION

Sex	No. of Patients	Percentage
Male	27	56%
Female	23	44%

Males were slightly more predominantly affected than females.

TABLE 3

SYMPTOM ANALYSIS

Leading Symptoms	
Breathlessness	35 (70%)
Palpitation	30(60%)
Weakness of limbs	7(14%)
Chest Pain	3(6%)
Syncope	1(2%)

Leading Symptom was breathlessness found in 70% of patients and 5% of patients were asymptomatic. No cases of Ortner's syndrome or Bronchiectasis were found among the patients with mitral stenosis.

TABLE 4

ETIOLOGICAL ANALYSIS

Etiology	No: (%)
RHD	35 (70)
CongenitalHD	2 (4)
Thyrotoxicosis	2 (4)
IHD	6 (12)
HTN	4 (8)
Cardiomyopathy	1 (2)

Rheumatic heart disease was the leading cause found in 70% of patients. The second commonest being ischemic heart disease was found in 12%.

TABLE 5

TYPE OF VALVULAR LESION

Total Number- 35 cases of RHD

Type of Valve Disease	No. of Patients
MS	18 (50%)
MR	5 (16%)
MS + MR	10 (28%)
MS+AR+AS	2 (6%)

Isolated Mitral Stenosis was the leading cause found in 50% of patients.

TABLE 6

SEVERITY OF MITRAL STENOSIS

Valve Area	No. of Cases	Percentage
> $1cm^2$	6	20%
< $1cm^2$	24	80%

Most of the cases were severe mitral stenosis with valve area <$1cm^2$.

TABLE 7

COMPLICATION

Complications	No. of Patients
CCF	30 (60%)
Thromboembolism	7 (14%)
I.E	5 (10%)

Congestive cardiac failure was the leading complication found in 60% of the patients. Whereas thromboembolism was found in only 14%.

TABLE 8

THROMBOEMBOLISM

Thromboembolism	Clot + No : (%)	Clot – No : (%)
Yes	5 (70)	2 (30)
No	2(4)	41(96)

Among the cases with thromboembolism almost 70% had left atrial clot with trans esophageal echo.

Peripheral Embolism – 1 (15%) Central – 6 (85%)

TABLE 9

SEX RATIO

Male	Female
5 (70%)	2 (30%)

Of the patients with embolism 70% were males.

TABLE 10

LEFT ATRIAL SIZE

LA Size	Total No.	> 4 cm	< 4cm
Permanent /Persistent AF	46	40	6
Paroxysmal AF	4	0	4

Left atrial size more than 4cms was found in almost 87% of patients.

DISCUSSION

An attempt has been made to study fifty cases of Atrial Fibrillation.

- Mohan Nair et al[1] concluded that atrial fibrillation is the most commonly sustained arrhythmia, being present in 0.4% of the overall population. This incidence is higher in countries with a high prevalence of rheumatic heart disease (RHD).

- Jeff S. Healey et al[14] Currently >2.2 million people in the United States have atrial fibrillation, and the annual incidence is approximately 19.2 per 1,000 person-years

AGE DISTRIBUTION

- In our study the age incidence is 21 -80 years with mean age of 43 years. 46% of study population were between 21-40yrs. 46% between the age group of 41-60 yrs & 8% in the age group of 61-80yrs.

- The mean age of the study population is 43 years in our study. Benjamin .O et al[4] and Abdou Elhandy et al[20] reported mean age as 58 & 67 in their respective studies. Maru[8] reported 136 Ethiopian cardiac outpatients with AF in whom the mean age was 41 years. Crijns et al[35] evaluated 127 patients, the average age of their patients was 60 years all of the above mentioned studies correlates with our study. AFFIRM[3] study reports mean age as 70 years, mean age in James et al study was 71 years. In the last two studies mentioned above age incidence is higher as most of their study population included were above 50 years as the cause of atrial fibrillation differed from our study.

MALE INCIDENCE

- 56% of our study population were male which correlates with AFFIRM[3] study were the incidence in males were 61%.Abdou Elhendy[20] reported male incidence as 66%, 52.63% male incidence was reported by Howard .S et al[5]. Framingham[4] heart study showed greater incidence of atrial fibrillation in males. Feinberg[31] also reported increased incidence in males.

ETIOLOGICAL ANALYSIS- RHD

- In our study out of 50 case 70% (35 cases) had rheumatic heart disease as the cause for developing Atrial Fibrillation.Bernand L.J et al[19] in their study concluded 70% of their study population had RHD as etiology. Maru[8] reported 136 Ethiopian cardiac outpatients with Atrial Fibrillation, in whom the mean age was 41 years, and the commonest cause was rheumatic heart disease (66%) ,Samuel levy et al[6] found RHD to cause Atrial Fibrillation in 33% of his study population.A.G Shaper[15] reported that higher incidence of RHD is common in tropical countries.
- Crijns et al[35] evaluated the characteristics of 127 patients of atrial fibrillation. Valvular heart disease was the cause in 24% of his study population.
- AFFIRM[3] study reports very low incidence of rheumatic heart disease in their study, similarly Abdou et al[20] reported low incidence. In western studies the most common cause of atrial fibrillation is hypertension as incidence of rheumatic fever is very low.
- Studies done in tropical countries show rheumatic heart disease as the major cause of atrial fibrillation which co-insides with our study result. Western studies report hypertension and coronary artery disease as the main cause, as the prevalence of rheumatic fever leading to rheumatic heart disease is not so common in developed countries.

ETIOLOGICAL ANALYSIS – Coronary Artery Disease

CAD is 2nd most common cause of atrial fibrillation with an incidence of 12% in our study. Samuel Levy et al[6] reported 16.6% of their study population had CAD as a cause for atrial fibrillation. In AFFIRM[3] study CAD was presented 38% of cases. Howard S. Friedman[5] reported 14% of cases. Abdou Elhendy et al[20] reported 34% of cases. According to Gregory lip et al[9] myocardial infarction complicates atrial fibrillation in 10-15% of cases. According to Aberg H[12] in his analysis of 463 patients with atrial fibrillation, coronary artery disease was the underlying cause in 23% of patients. The above mentioned studies correlate with our study regarding coronary artery disease as risk factor, though in some studies the major causative factor was hypertension.

HYPERTENSION

In our study the incidence of systemic hypertension was 8%.

Crijns et al[35] evaluated the characteristics of 127 patients with atrial fibrillation were he found . hypertension was the etiological factor in 16% of cases.

Abergh[12] in his analysis of 463 cases of atrial fibrillation noticed 34% of cases had systemic hypertension.

According to Framingham[4] study, hypertension accounted for about half of the cases.

Hypertension was present in 71% of cases in AFFIRM[3] study. Abdou Elhendy et al[20] reported incidence of systemic hypertension as 51% in their study. The incidence of Hypertension in our study is much lesser than other trials.

SYMPTOM ANALYSIS

- Most of the patients in our study population presented to emergency department with shortness of breath as their main complaint 70% (35 patients) with overlapping of other symptoms. Palpitation was present in 30 patients (60%) .Other complaints in decreasing order were chest pain 6% (3 cases),weakness of limbs in 7 cases (14%) ,syncope in 1 case (2%), asymptomatic 5cases (10%).
- . In the Canadian Registry of Atrial Fibrillation (CARAF)[7], only 21% of patients were asymptomatic on presentation. Among the 79% of patients with symptoms, palpitations occurred in 50%, chest pain and fatigue in more than 25% and dizziness, presyncope or syncope in about 25%.As in our study palpitation was present in 60% of study population , chest pain present in 6%of cases.
- Benjamin O. Koenig et al[4] reported the following data in his study

Presenting symptoms (%)	
Palpitations	57 (85)
Chest pain	15 (22)
Dizziness	12 (18)
Shortness of breath	12 (18)
Weakness	5 (7)

- The percentage of patients with shortness of breath varies from our study ,other symptoms correlates. The number of patients included in their study was very high.

Cardioversion

15% of our study population were haemodynamically unstable and underwent emergency cardioversion.

Benjamin et al[4] in is study disclosed that emergency cardioversion was needed in 17% of his study population. 10% of cases who were haemodynamically unstable in the study done by Paul et al[38] were cardioverted.

Farhat Khairallah et al[23] did Electrical cardioversion in 10% of their study population.

The above mentioned studies showed more or less similar percentage of patients who needed emergency cardioversion.

Common Valvular Lesion in patients with RHD

In our study MS+MR was present in 10 cases 28%, MS in 18cases, 50%, MR in 5 cases 16%,MR+AS+AR in 2 cases, 15%.

The AFFIRM[3] Investigators found that mitral regurgitation was present in 15% of their cases

According to R Arora et al[16] in their study of 2500 cases MS+MR was presented in 30%, MS was present in 38% of cases. Cihangir Kazmay et al[21] reported MS in 70% of cases and MR in 30%

Complications:

In our study the most common complication is congestive cardiac failure 60% (30 cases). AFFIRM[3] suggests that 32% of their patients had decreased left ventricular function. According to O.T Samani[26] congestive cardiac failure was presented in 64.9% of cases with atrial fibrillation.

The common thought of increased incidence of thromboemobolic episode in atrial fibrillation is proven wrong, as per our study left ventricular dysfunction leading to heart failure is the most common complication.

In our study 7 patients (14%) had thromboembolic episode, 6 of them being cerebral embolism which corresponds with cabin HS , et al[13] who reported cerebral embolism in 85% of cases and peripheral embolism in 15% of cases. In our study 70% of patients with clinical features of embolism had left atrial clot and only 4% without embolism had it.Goswamy KC et al[41] reported 26% incidence of LA clot in their study on 200cases of AF. They concluded that large left atrium & spontaneous echo contrast were associated with higher risk for clot formation. Rao[42] AS et al in their study on 120 patients with mitral stenosis and AF by TEE observed LA thrombus in 34 patients (25%) compared with 21(16%) by TTE. In the study most left atrial appendage clots were missed by TTE . In our

study the incidence of LA clots by TEE was 7(14%) out of the fifty patients with AF. Among the patients 6 had rheumatic MS and total number cases of AF with MS was 30. The incidence being 20% which correlated with the above mentioned study. By TTE only 5 cases were detected to have clot. Yadav R et al[43] in their study on 200 patients with MS reported 25% incidence for LA appendage clot and risk being higher in patients with older age, larger left atrium and atrial fibrillation.

Left Atrial Dilatation

87% (40 patients) of our study population had left atrial dilatation which correlates with AFFIRM[3] study where left atrial dilatation was present in 67% of their study population.

Study done by Zabalgoitia et al on 786 patients found out left atrial dilatation was present in 67% of cases, Adbou Elhendy et al[20] in their study reported 81% of cases to have left atrial dilatation were mild enlargement observed in (41%) and marked enlargement seen in (40%) of his study population.All the studies show high incidence of LA dilatation as per our study. LA size more than 4cm was found to be associated with permanent atrial fibrillation.

CONCLUSIONS

1. AF is seen more commonly in men.
2. Mean age of the study population was 43 years.
3. Most common cause was RHD with mitral valve involvement.
4. Common presenting symptom was dyspnoea.
5. 15% of the patients with AF had significant haemodynamic instability requiring cardioversion.
6. CCF was the most common complication in 60% of patients and CVA in 14% of patients.
7. Echo revealed LA dilatation in 80% of cases.
8. LA clots were present in 14% of all cases and in 70% of pts with embolism.
9. Left atrial size more than 4cms was found to be related to permanent or persistent atrial fibrillation.

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PROFORMA

CLINICAL PROFILE OF ATRIAL FIBRILLATION

NAME	:	
AGE / SEX	:	
I.P. No	:	
Complaints on Admission	:	Duration:
Shortness of Breath	:	
Palpitation	:	
Chest Pain	:	
Syncope	:	
Weakness of limbs	:	
Others	:	(specified)
Past History	:	Duration :
DM	:	
HTN	:	
CAD	:	
Hyper lipedemia	:	
COPD/BA	:	
RHD	:	

General Examination :

Consciousness :

Pallor, Ictirus, Clubing, Cyanosis, Lymphadenopathy,

Pedal Edema

JVP :

Vitals

Pulse rate :

Pulse deficit :

BP :

Temperature :

Respiratory Rate :

SpO_2 :

Examination of systems:

CVS :

RS :

CNS :

Abdomen :

Haemodynamically Unstable Yes / No

Labs :

CBC :

RFT :

LFT :

Sr. Electrolytes :

Free T_4 and TSH :

ECG :

ECHO :

Valvular abnormality

LA dilatation

LA clot / Vegetation

X-Ray Chest :

Cardiomegaly Yes / No

Treatment :

Outcome :

MASTER CHART

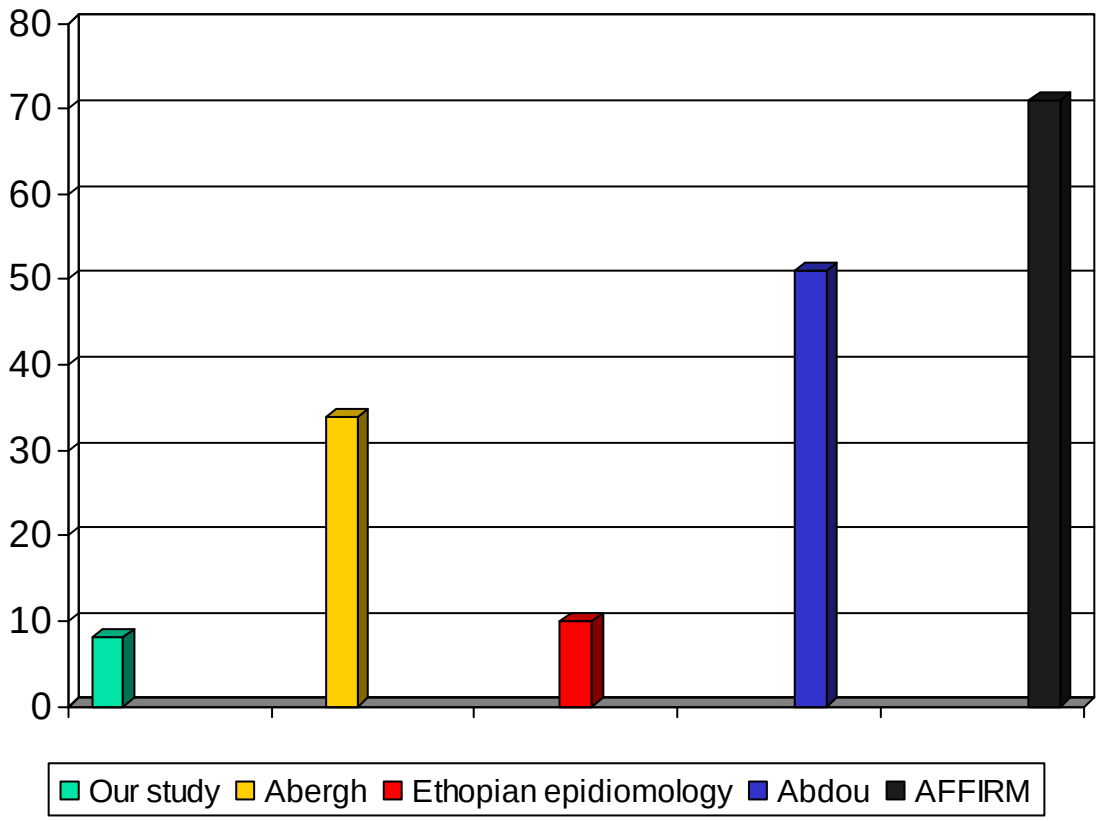
SL. No.	Name	Age	Sex	Etiology	Type of Valve Disease	Valve Area (Cm^2)	Cardio-version	CCF	Thrombo-embolism	TEE		H/o RF	I.E.	CXR (Cardio-megaly)
										LA size	LA clot			
1.	Munian	40	M	RHD	MS	<1	-	+	+	5	+	+	-	-
2.	Kamatchi	30	F	RHD	MS	<1	-	+	-	4.8	-	+	-	-
3.	Raja	28	M	RHD	MS	<1	-	+	-	4.3	-	+	-	-
4.	Ramasamy	43	M	RHD	MS	>1	+	+	-	4	+	-	-	-
5.	Arumugam	48	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+
6.	Meena	20	F	CHD	-	-	-	+	-	4.7	-	-	-	-
7.	Veerasamy	60	M	IHD	-	-	-	+	-	4	-	-	-	+
8.	Muniamma	51	F	HTN	-	-	-	-	-	3.8	-	-	-	+
9.	Ramasamy	48	M	RHD	MS+AS+AR	<1	+	+	+	5	+	+	-	+
10.	Kanniammal	36	F	Thy	-	-	-	-	-	3.5	-	-	-	-
11.	Kathiravan	47	M	RHD	MS+AS+AR	<1	+	+	+	5	+	+	-	+
12.	Karuppan	48	M	RHD	MS+AS+AR	<1	+	+	+	5	+	+	-	+
13.	Usha	36	F	Thy	-	-	-	-	-	3.5	-	-	-	-
14.	Ethiraj	43	M	RHD	MS	<1	-	+	+	5	+	+	-	-
15.	Dhanalakshmi	18	F	RHD	MS	<1	-	+	-	4.2	-	+	-	-
16.	Samy	28	M	RHD	MS	<1	-	+	-	4.3	-	+	-	-
17.	Kandan	40	M	RHD	MS	>1	+	+	-	4	+	-	-	-
18.	Arul	48	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+
19.	Roja	30	F	RHD	MS	<1	-	+	-	4.1	-	-	-	-
20.	Basha	61	M	DCM	-	-	-	+	-	4	-	-	-	+
21.	Subbulakshmi	30	F	RHD	MS	<1	-	+	-	4.5	-	+	-	-
22.	Manickam	29	M	RHD	MS	<1	-	+	-	4.3	-	+	-	-
23.	Nandagopal	43	M	RHD	MS	>1	+	+	-	4	+	-	-	-

24.	Joseph	48	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+
25.	Jothi	20	F	RHD	MS	<1	-	+	-	4.9	-	-	-	-
26.	Maahesh	47	M	RHD	MS	<1	-	+	+	5	+	+	-	-
27.	Vanitha	32	F	RHD	MS	<1	-	+	-	4.1	-	+	-	-
28.	Suresh	28	M	RHD	MS	<1	-	+	-	4.3	-	+	-	-
29.	Prakash	43	M	RHD	MS	>1	+	+	-	4	+	-	-	-
30.	Saravanan	52	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+
31.	Sri Devi	20	F	RHD	MS	<1	-	+	-	4.8	-	-	-	-
32.	Aruchamy	62	M	IHD	-	-	-	+	-	4	-	-	-	+
33.	Kandhimathi	55	F	HTN	-	-	-	-	-	3.8	-	-	-	+
34.	Kulandaisamy	45	M	RHD	MS	>1	+	+	-	4	+	-	-	-
35.	Jayapal	49	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+
36.	Ezhilarasi	20	F	RHD	MS	<1	-	+	-	4.6	-	-	-	-
37.	Kumanaraja	65	M	IHD	-	-	-	+	-	4	-	-	-	+
38.	Delli	51	M	RHD	MS+AS+MR	<1	+	+	+	5	+	+	-	+
39.	Raji	30	F	Thy	-	-	-	-	-	3.5	-	-	-	-
40.	Rajaram	40	M	RHD	MS	<1	-	+	+	5	+	+	-	-
41.	Jothiga	31	F	RHD	MS	<1	-	+	-	4.2	-	+	-	-
42.	Dass	36	M	RHD	MS	<1	-	+	-	4.3	-	+	-	-
43.	Thotti Jaya	43	M	RHD	MS	>1	+	+	-	4	+	-	-	-
44.	Pazhanivel	48	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+
45.	Vijaya	20	F	RHD	MS	<1	-	+	-	4.5	-	-	-	-
46.	Kondaiah	63	M	IHD	-	-	-	+	-	4	-	-	-	+
47.	Kanimozhi	30	F	RHD	MS	<1	-	+	-	4.4	-	+	-	-
48.	Praveen	34	M	RHD	MS	<1	-	+	-	4.3	-	+	-	-
49.	Mustafa	43	M	RHD	MS	>1	+	+	-	4	+	-	-	-
50.	Raghavan	53	M	RHD	MS+ MR	>1	-	-	-	4	-	-	-	+

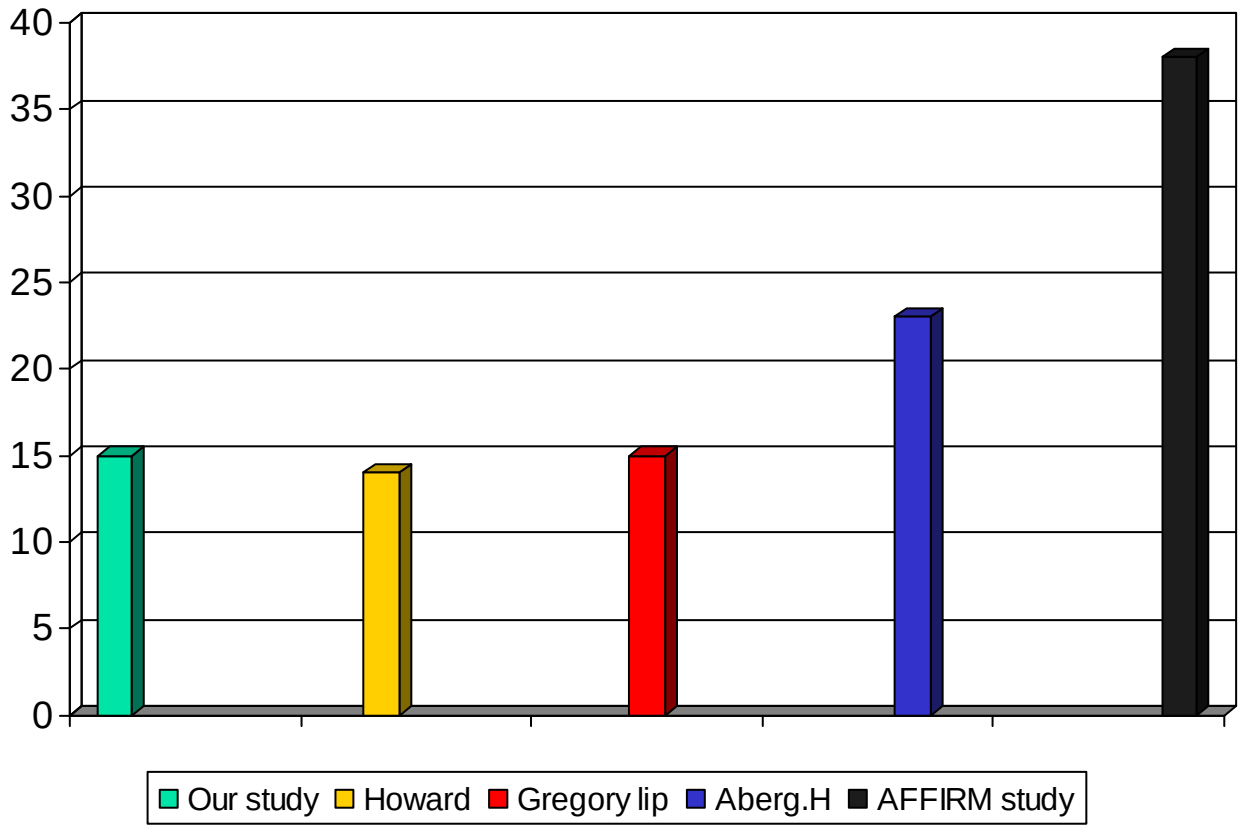
KEY TO MASTER CHART

TEE	:	Transesophageal Echo
RHD	:	Rheumatic Heart Disease
DCM	:	Dilated Cardiomyopathy
LA	:	Left Atrial
RF	:	Rheumatic Fever
IHD	:	Ischemic Heat Disease
Thy	:	Thyrotoxicosis
HTN	:	Hypertension
CHD	:	Congenital Heart Disease
MS + MR	:	Mitral Stenosis + Mitral Regurgitation
AR	:	Aortic Regurgitation

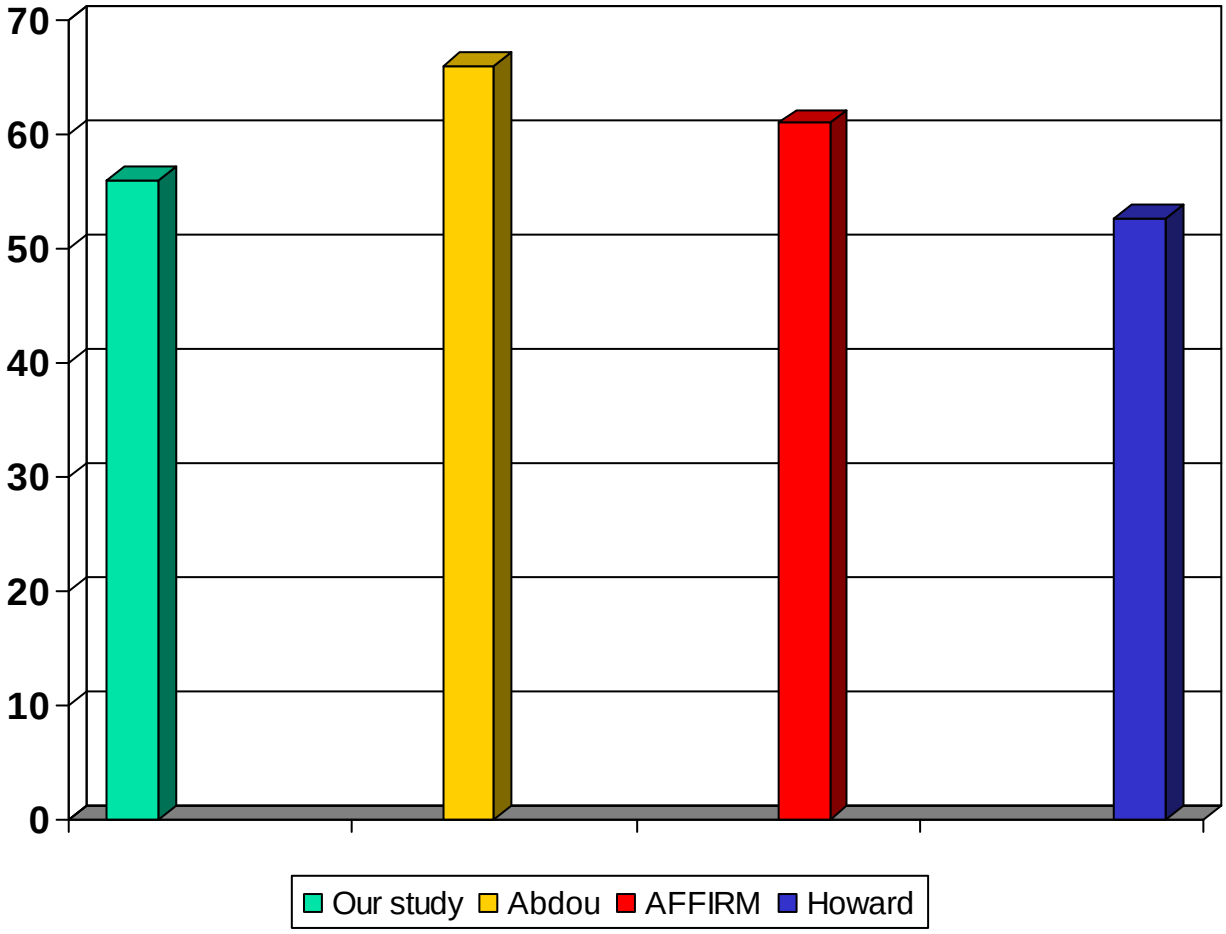
AETIOLOGICAL ANALYSIS - HYPERTENSION



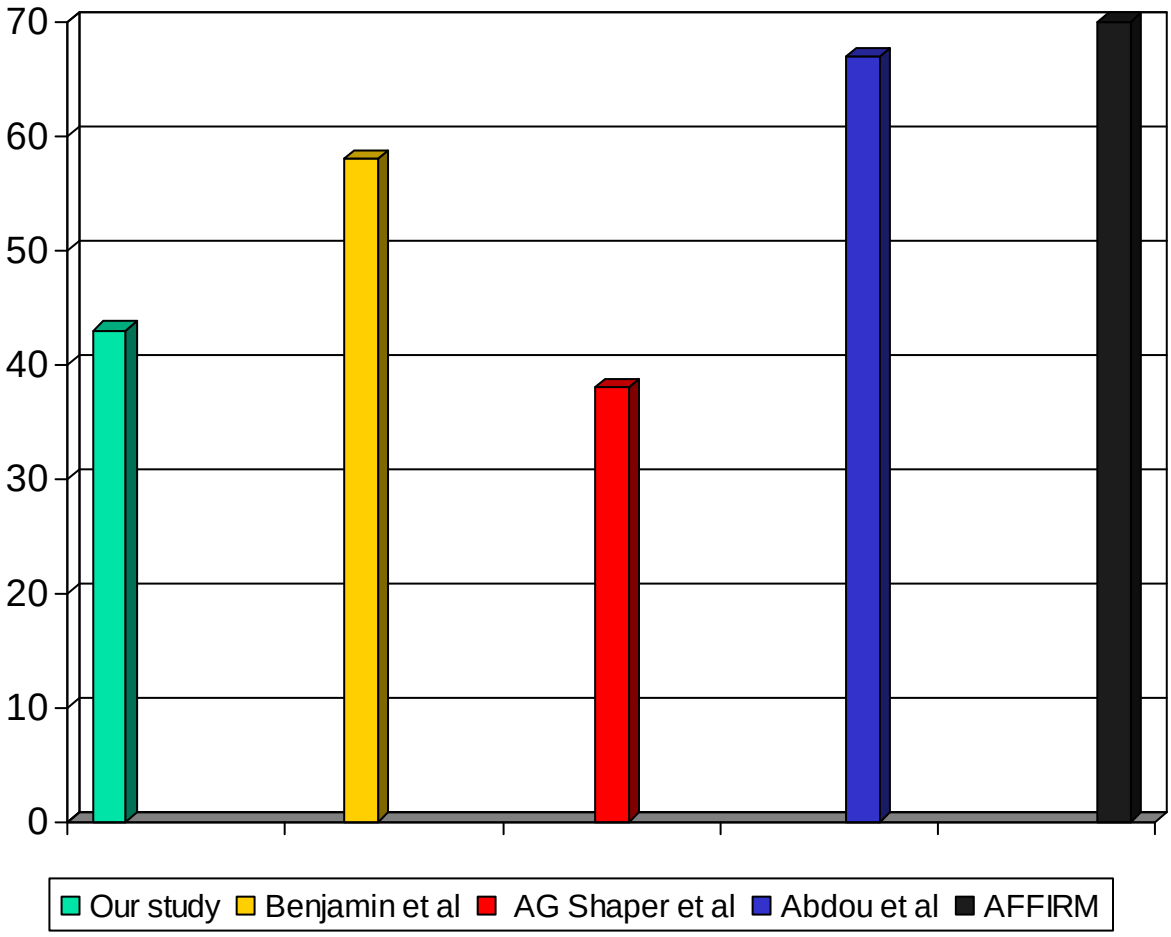
AETIOLOGICAL ANALYSIS – CORONARY ARTERY DISEASE



SEX : MALE INCIDENCE



MEAN AGE


70
60
50
40
30
20
10
0
Our study
Benjamin et al
AG Shaper et al
Abdou et al
AFFIRM

11-20	21-30	31-40	41-50	51-60	61-70
8	7	8	15	8	4

Male	Female
54%	46%

Breathlessn	Palpitaion	Weakness	Chest Pain	Syncope
70%	60%	14%	6%	2%

RHD	CHD	Thyrotoxico	IHD	HTN	Cardiomyopathy
70%	4%	4%	12%	8%	2%

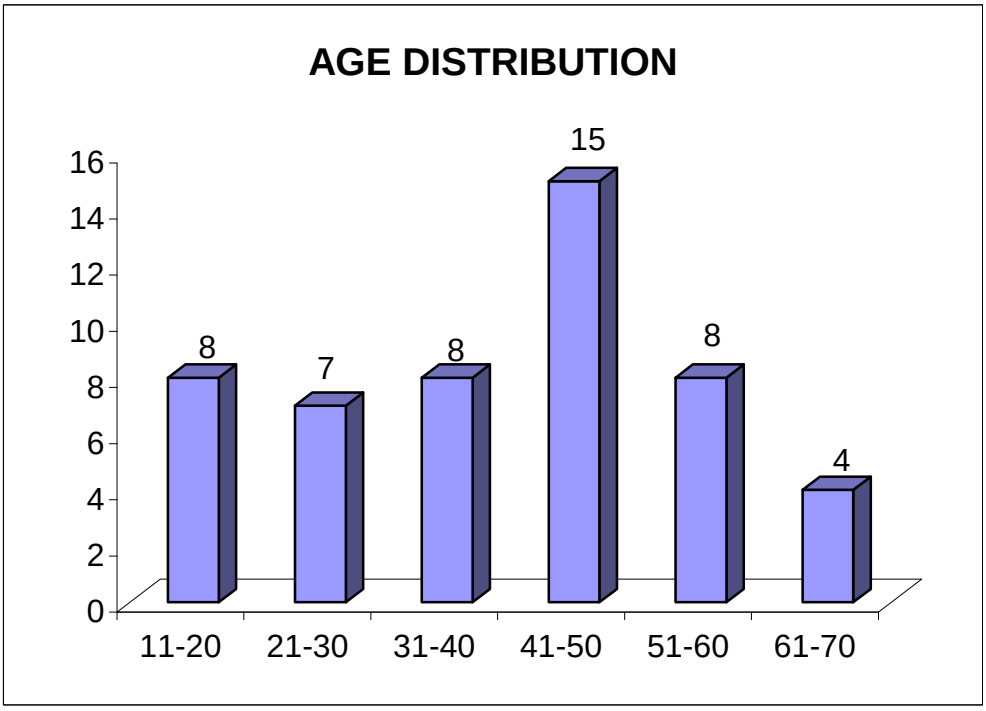
MS	MR	MS+MR	MS+AR+AS
50%	16%	28%	6%

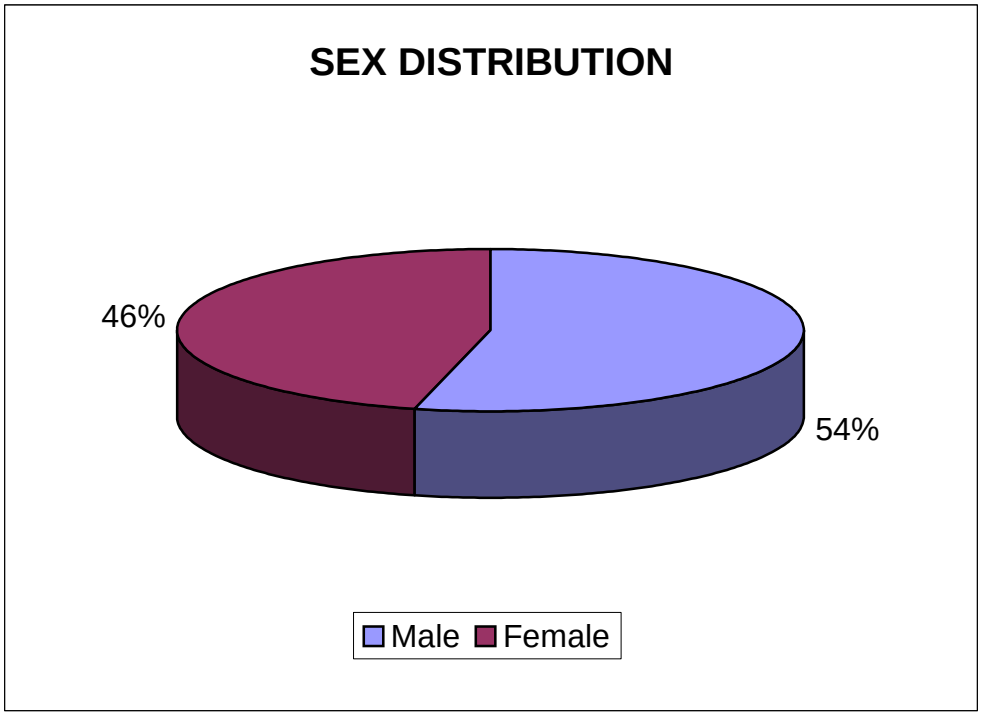
CCF	Thromboer	I.E.
60%	14%	10%

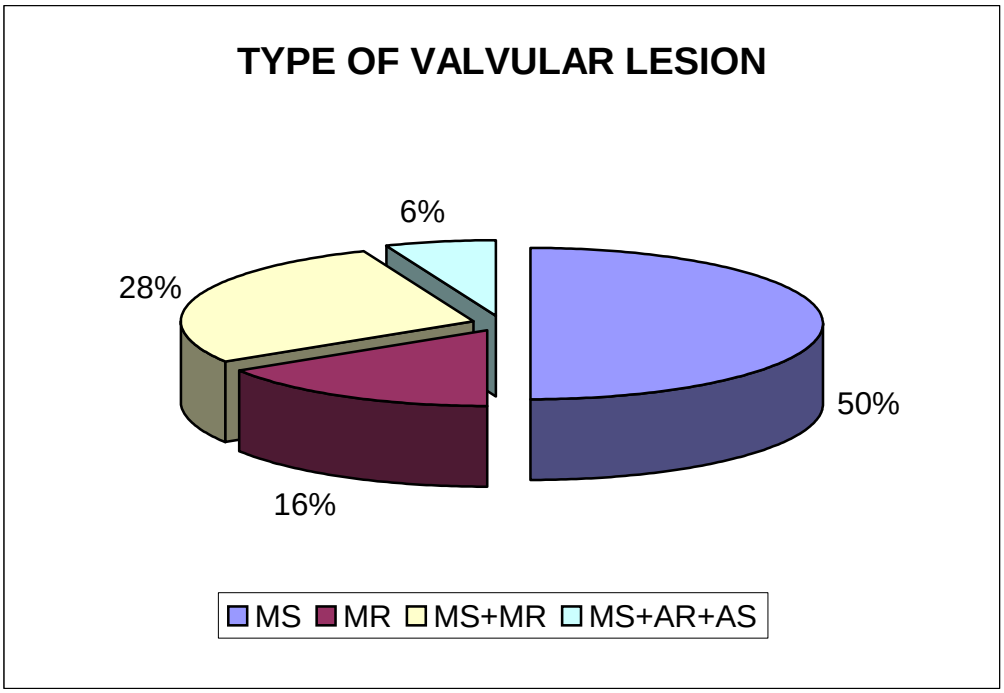
	Clot +ve	Clot -ve
Yes	70%	30%
No	4%	96%

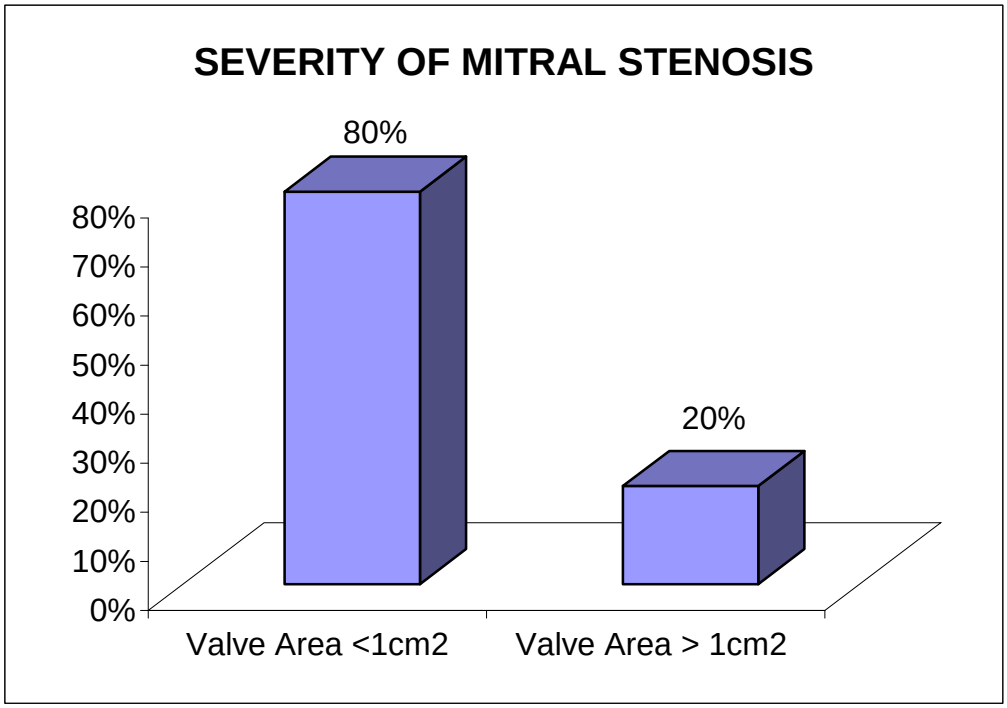
Valve Area	Valve Area > $1cm^2$
80%	20%

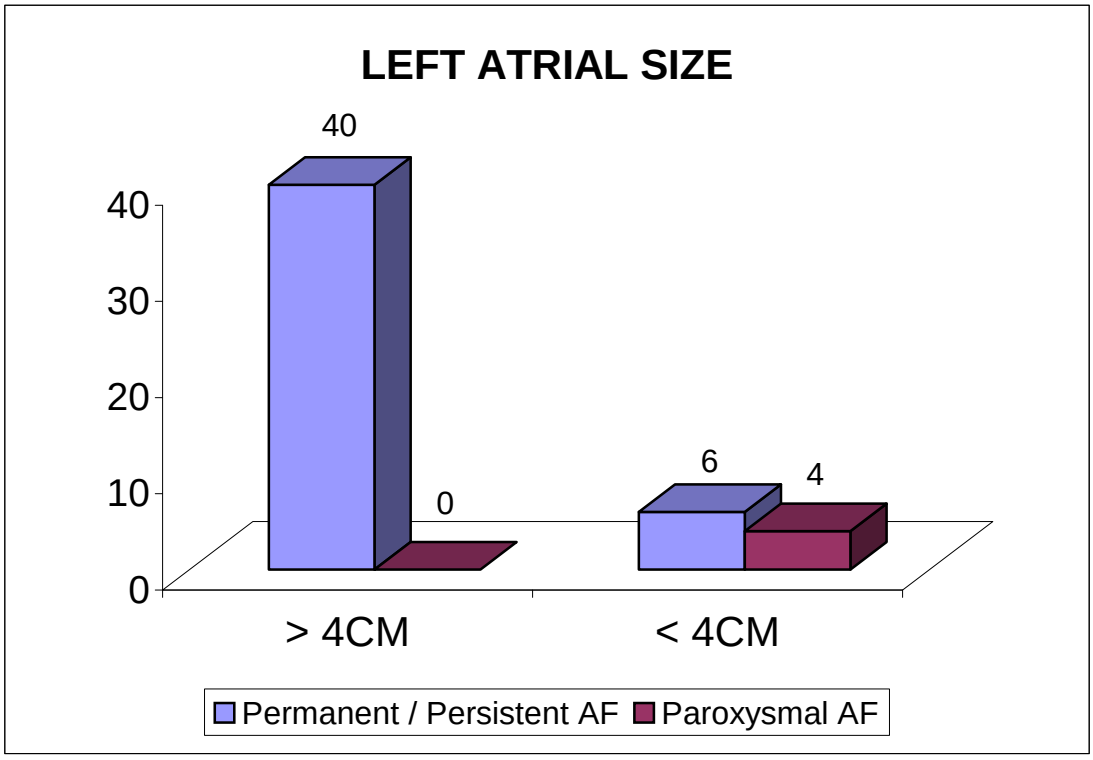
	> 4CM	< 4CM
Permanent	40	6
Paroxysma	0	4

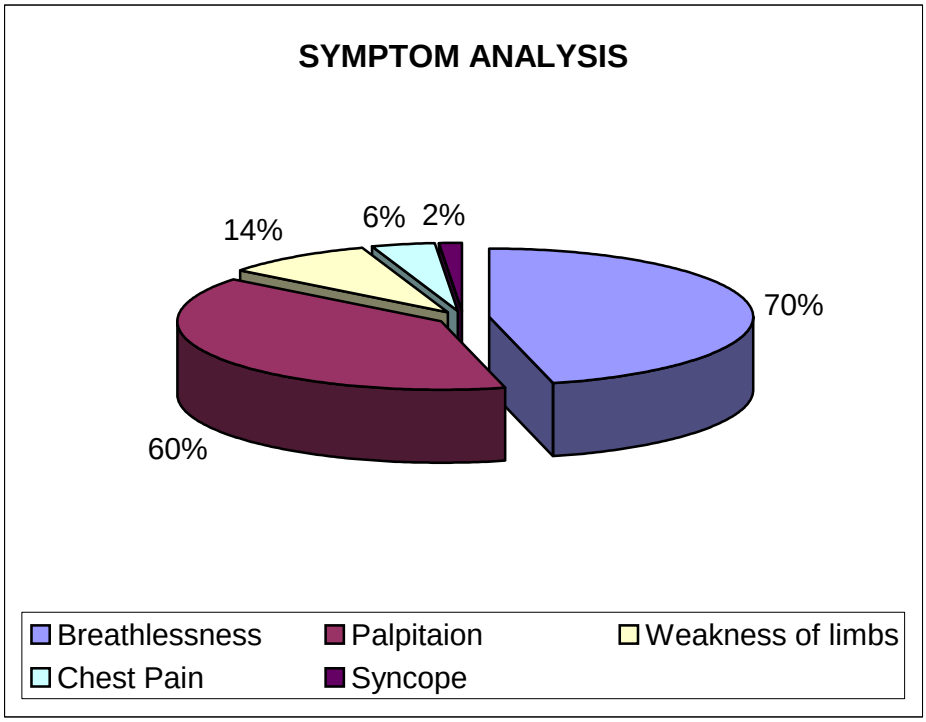

AGE DISTRIBUTION
16
14
12
10
8
6
4
2
0
8
7
8
15
8
4
11-20
21-30
31-40
41-50
51-60
61-70

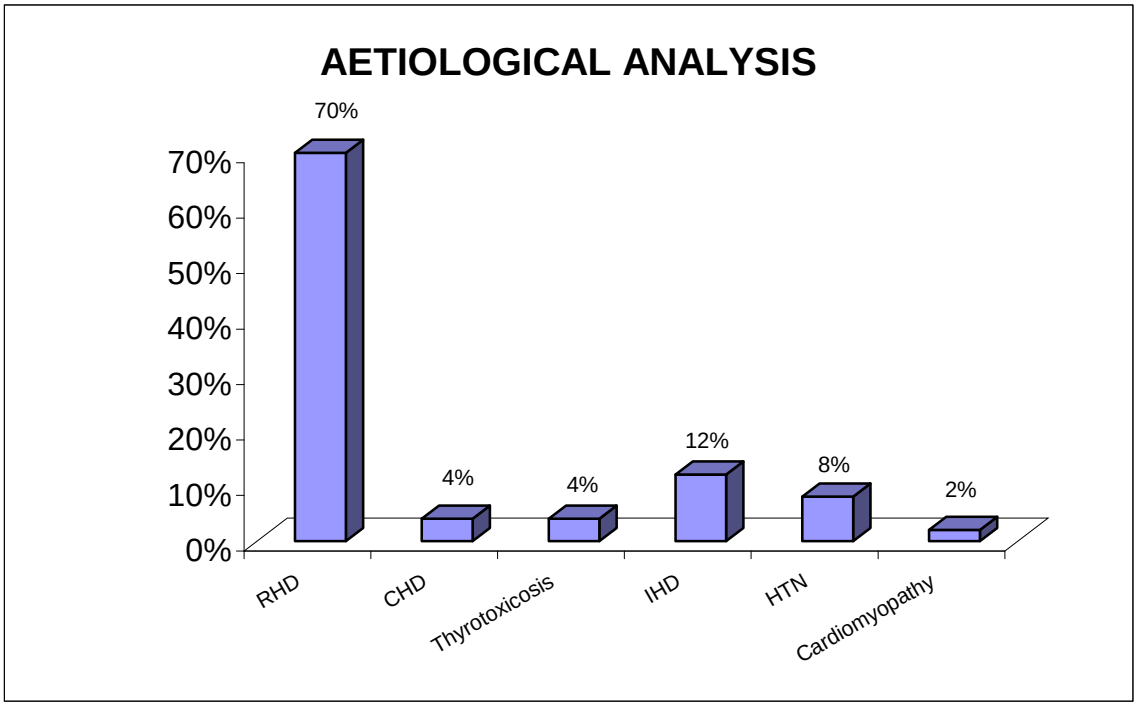

SEX DISTRIBUTION
46%
54%
Male
Female

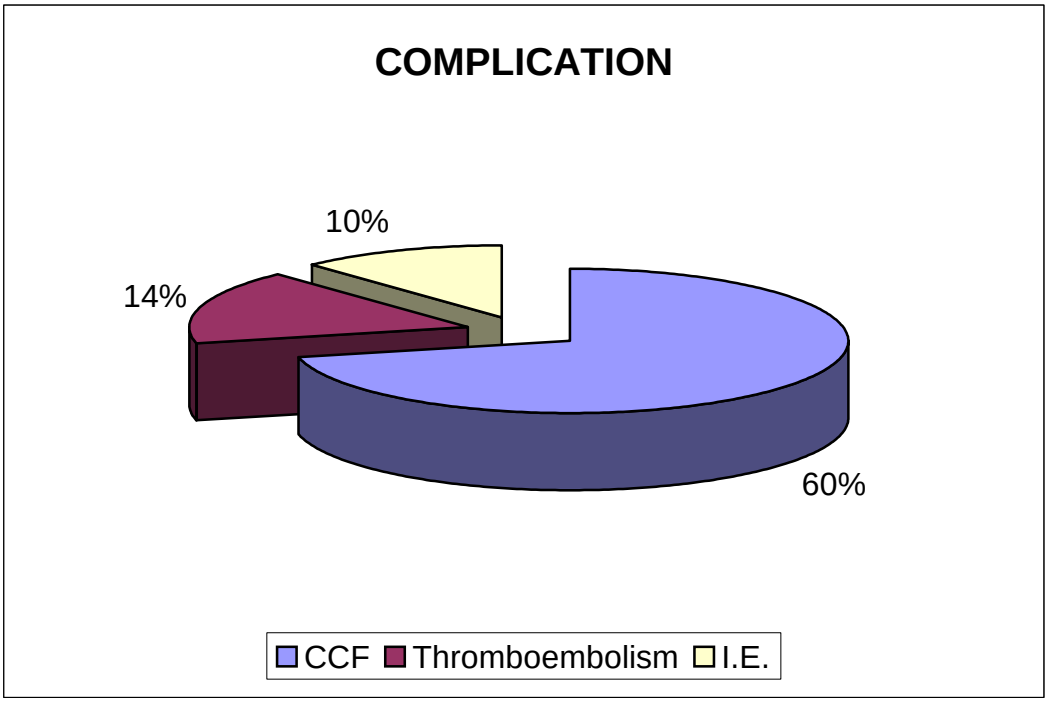

TYPE OF VALVULAR LESION
6%
28%
50%
16%
MS
MR
MS+MR
MS+AR+AS

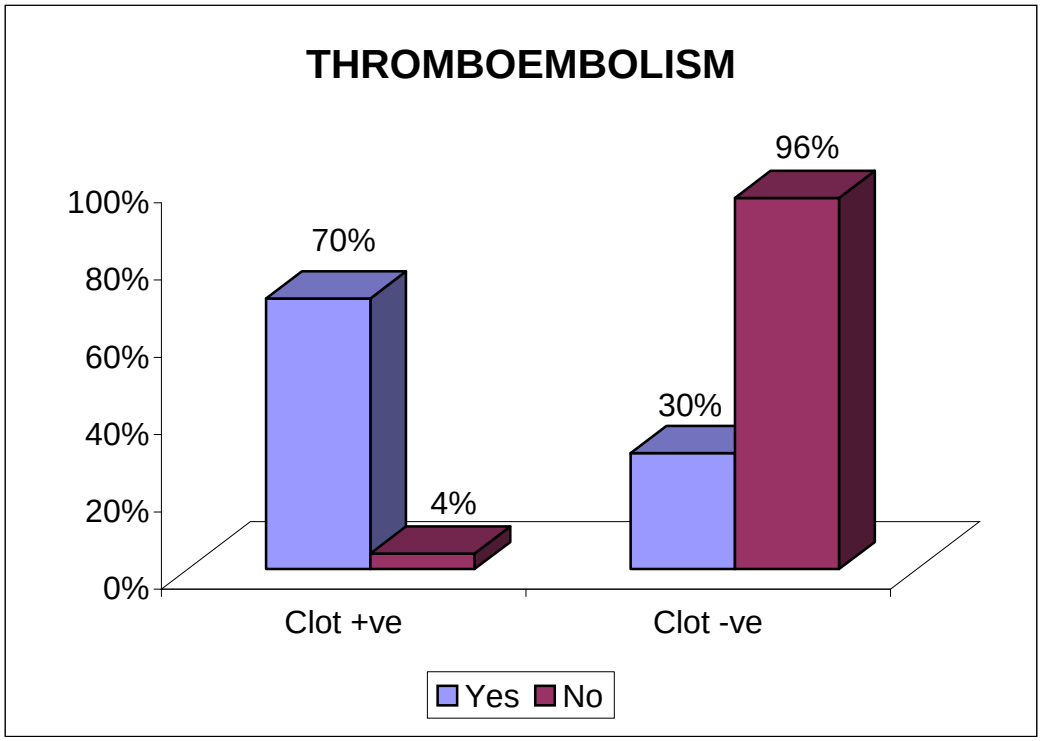

SEVERITY OF MITRAL STENOSIS
80%
20%
80%
70%
60%
50%
40%
30%
20%
10%
0%
Valve Area <1cm2
Valve Area > 1cm2


LEFT ATRIAL SIZE
40
0
6
4
40
30
20
10
0
> 4CM
< 4CM
Permanent / Persistent AF
Paroxysmal AF


SYMPTOM ANALYSIS
14%
6%
2%
70%
60%
Breathlessness
Palpitaion
Weakness of limbs
Chest Pain
Syncope


AETIOLOGICAL ANALYSIS
70%
70%
60%
50%
40%
30%
20%
10%
0%
4%
4%
12%
8%
2%
RHD
CHD
Thyrotoxicosis
IHD
HTN
Cardiomyopathy


COMPLICATION
10%
14%
60%
CCF
Thromboembolism
I.E.


THROMBOEMBOLISM
100%
80%
60%
40%
20%
0%
70%
4%
30%
96%
Clot +ve
Clot -ve
Yes
No